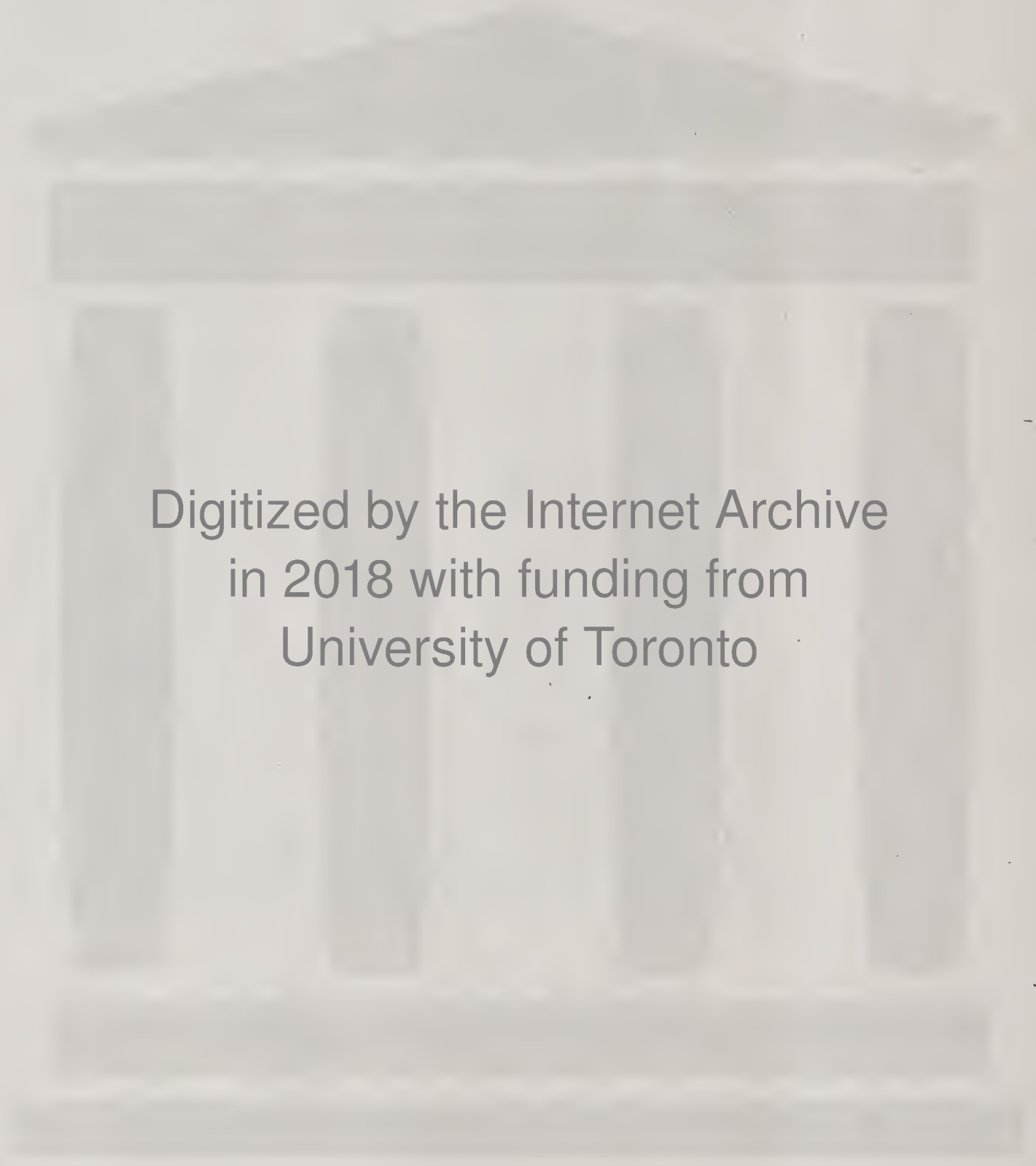




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THE UNIVERSITY OF TORONTO MEDICAL JOURNAL

(An Undergraduate Publication)

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Volume IX

TORONTO, MAY, 1932

Number 7

EDITORIAL

IN THIS ISSUE.

In this issue we print a very interesting historical article written by Dr. J. H. Elliott, who has been recently appointed to the position of Professor of the History of Medicine. This information, gleaned from old minute books of the Medical Society, which Dr. Elliott very fortunately has obtained, deals with the early organization and events of the Medical Society of the University of Toronto.

The Medical Society Executive is anxious to learn the whereabouts of the Minute Books of the Society covering the period from 1896-1922. Dr. Elliott has obtained the Minute Books from 1876 to 1896, and with this excellent start, an effort is being made to

complete the records of the Medical Society. Should any reader know where these records may be found, it would be appreciated if they would communicate with Dr. Elliott, or with the Secretary of the Medical Society, Room 107, Anatomy Building.

MEDICAL STATISTICS.

How often does one read in medical publications a statement such as this—such and such a treatment produced a cure in 5 out of 7 cases — an efficacy of 71.4%. This may be an extreme example, but it serves to illustrate some fallacies which at times are seen in medical writings. In the first place, very often the number of

cases are too few to justify any exact and definite conclusions being drawn. In the second place, the calculation of the above percentage from the available data is absurd, since 71.4% means 714 cases out of 1,000.

Another fault which appears in the best of medical work is the incomplete valuation of all the variables in a particular instance. This has been brought out very forcibly in connection with the many and varied treatments introduced for pneumonia. A certain worker, working during a certain winter season, finds that, by using a certain method of treatment, he experiences a low mortality, and immediately the medical profession is informed of the wonderful cure for pneumonia. However, the next winter, the same worker and others use the same treatment and find that a much higher mortality results. What is the explanation? Has the treatment become miraculously ineffective over the summer? No! A variable has been neglected—pneumonia may vary in its virulence and mortality from season to season, with or without treatment.

Since medicine, of all the sciences, shows so many variables, it well behoves every member of the medical profession to recognize the limitations which a certain set of data may show, and appreciate the possibility of many variables entering into any field of medical endeavour—in short, be critical, but yet open-minded.

GRADUATING YEAR.

Some members of the graduating year will no doubt wish to continue their subscriptions to the Journal.

Many have complete sets of six and seven volumes and realizing that the value will be the same, or more so, after graduation, will continue to subscribe.

Volume I. of the Journal has long been unavailable for purchase; even the Journal library needs these numbers to make it complete. An opportunity is here presented for philanthropically minded graduates to make what would be an appreciated donation to the Medical Society. We understand that the Academy of Medicine is also desirous of obtaining copies of the first volume. Should copies of the first numbers of the University of Toronto Medical Journal be for sale, the Journal Staff would be glad to hear of it.

THANK YOU.

As the last chapter is written and the printer's rule laid aside, the Editor would voice appreciation to those who have made possible the issues of Volume IX.

The editorial work of the Journal—the writing, selecting, proof-reading—has been shared by each member of the staff. The advertising, financing and distribution have been efficiently handled by the Managing Editor and his assistants.

We would thank the members of the faculty, graduates, undergraduates, who have contributed of their time, interest, and thoughtful research in articles. Dr. E. S. Ryerson, the Journal Advisor, has helped the editorial “lame dog” over many a journalistic stile.

The appointment of Mr. R. B. Kerr to the Editorship ensures the success of Volume X.

The Early Records of the University of Toronto Medical Society.*

By J. H. ELLIOTT, M.B., F.R.C.P. (Can.)

The University of Toronto Medical Society dates back to 1878. On December 9 and December 11 of that year meetings were held "to receive report of Committee appointed to draft a Constitution for the proposed Literary Society." The draft, presented by Mr. G. B. Smith, was prepared by Mr. H. Wilberforce Aikins, B.A., who received a vote of thanks for his work.

The Constitution as finally adopted comprised three Articles, with sub-headings.

Art. I.

(1) The Society shall be called *The Toronto School of Medicine Literary and Debating Society*.

(2) It shall consist of former and present students of The Toronto School of Medicine.

(3) The objects shall be the encouragement among the members (a) of intellectual pursuits, of discussions, essays and readings. (b) no discussions upon party politics or controversial points shall be allowed.

(4) The patrons shall be the members of the Faculty of the School.

Art. II. This relates to membership—ordinary, life and honorary. Nominations for membership were to be made at one meeting and to be declared elected at the next if no ballot were demanded. If a ballot were demanded, a three-quarter vote was necessary to

elect. No member could vote or hold office unless his fees were paid.

Art. III. This relates to the officers.

A President was elected for the remainder of the term, with G. B. Smith, '80, as Vice-President, George S. Bingham, '81, Secretary, Mr. Pettit Treasurer, and a Council composed of Rev. J. McCarroll, 4th year, J. T. Dickson, 3rd year, Hugh Watt, 2nd year, J. T. Duncan, 1st year. In addition to those of the officers the names appearing on the record of these meetings for organization are H. W. Aikins, B.A., Mr. Milne, Mr. J. Bell, Charles McDonald, '81, Mr. W. H. Poole, W. C. Edmondson, '81, C. J. Hamilton, '80, J. G. Hyde, '80, Hamilton Meikle, H. R. Elliott, '81, R. M. Fisher, '81, Mr. Kenning.

The minutes for 1878 and 1879, with the copy of the Constitution, are in the writing of George S. Bingham, '81. Space was left for the bylaws, but unfortunately these have not been written in. Following the organization meetings the new Literary and Debating Society promptly proceeded to work. The "Programme of Entertainment" at the meeting of January 16, 1879, was as follows:

"Peter H. Bryce, M.A., read an essay on "Man." Rev. J. M. McCarroll read "The Soldier's Pardon." Next in order came the debate, the question being, "Is it advisable that the standard of the Matriculation of the College of Physicians and Surgeons of Ontario should be raised?"

"For the affirmative Messrs. J. Ferguson (leader), G. S. Bingham and H. W. Aikins, B.A., spoke. For the negative Messrs C. J. Hamilton (leader), Hugh Watt and J. H. Duncan. The chairman reviewed the debate and gave his decision in favor of the affirmative."

*This sketch of the early days of the Medical Society of the University of Toronto has been made from the Minute Book covering the years 1878-1896. Two pages of the minutes of 1896 and four pages of 1893-4, are missing, but otherwise the record is complete. When a new minute book was necessary, in 1896, the older volume was carefully preserved by Dr. J. C. Lindsay ('98), of London, Ont. It will be deposited in the collection of minute books of medical organizations in the Academy of Medicine where we hope all other such records may be placed.

Under business we read: "Moved by Mr. Bryce, seconded by Mr. C. J. Hamilton, 'That on account of Mrs. Scott Siddon's Readings the next regular meeting be postponed from Thursday evening, January 30th inst., to Friday evening, January 31st, inst.'"

On motion of Mr. H. W. Aikins and Mr. John Ferguson ('80) the meeting adjourned.

There is no list of members, but at the next meeting, January 31, we find Mr. H. Y. Baldwin, G. W. Clendenan and Hamilton Meikle nominated for membership. Under "Entertainment" the following items appear: "Mr. J. Ferguson read an essay on Italy. Mr. L. M. Sweatnam recited "Tell, to His Native Hills." Mr. Mennie sang "Gathering up the shells from the seashore," and for encore, "Three thousand miles away." Then followed the debate upon the question, "Resolved that ladies should enter the medical profession." Messrs. Milne (leader), Bryce, M.A., and Petit upheld the resolution, while Messrs. MacKechnie (leader), W. H. Aikins and Edmondson spoke against it. After summing up the arguments advanced pro and con, the President as Chairman decided in favor of the affirmative."

On February 13 in the absence of the President and Vice-President, Mr. H. W. Aikins presided. "The entertainment consisted of a reading, 'The Country Squire,' by Mr. Bell. In lieu of the essay Mr. Milne kindly occupied the time by reading a selection from Longfellow's Legend of Hiawatha. For debate the subject was: "Resolved that the use of tobacco should be discountenanced by the medical profession." The speakers for the affirmative were Messrs. J. T. Duncan (leader), Watt and Willmott; for the negative, Messrs. Poole (leader), Thompson and Ferguson. The chairman, after summing up the arguments advanced by both sides, decided in favor of the affirmative. The meeting adjourned on motion of Messrs. Watt and H. W. Aikins."

The last meeting of the year was held March 3, 1879, when the general

committee concluded its report "by hoping that the Society would soon possess a large and well-assorted library of medical works for the use of its members and the students in general." "Mr. McKechnie, the Treasurer pro tem, proceeded to collect from those present who had not already paid their fees." The officers elected for the ensuing year were:

President—H. W. Aikins.

Vice-President—W. H. Aikins.

Secretary—G. S. Bingham.

Treasurer—J. Bell.

Committee—Messrs. Edmondson, Bryce, D. W. Montgomery and Poole.

The meeting was then brought to a close by the members joining hands and singing heartily a few verses of "Auld Lang Syne."

In the session of 1879-1880 five meetings were held with H. W. Aikins as President. Among the items on the various programmes we find vocal solos by Mr. J. G. Mennie, J. Anderson, readings by L. M. Sweatnam, G. L. Milne, J. Bell, W. H. Johnson.

Essays—The Future of Canada, by P. H. Bryce, M.A.

The Ancient Literature of the Hindoos by J. T. Duncan.

The Jewish Race by J. Ferguson.

There were two subjects for debate:

(1) "Resolved that the present curriculum in Medicine of Toronto University is calculated to advance the best interests of Medical Science in connection with the University and the Toronto School of Medicine." The affirmative was supported by J. H. Duncan and W. C. Edmondson. The negative by J. Ferguson and Hugh Watt. (2) "That Canadian independence is preferable to British Connection." One hundred copies of the Constitution were ordered printed. The officers elected included J. T. Duncan, W. C. Edmondson, W. H. Johnson, W. A. Ross, A. D. Watson, J. Bell, and S. Bingham. Among the members elected were W. H. Montague, W. A. Ross, A. D. Watson, R. M. Coulter, J. B. Whitely, H. R. Elliott, —. McCracken, W. Gilpin, E. G. He-

wish, W. H. Johnson, W. J. Cross and W. E. Hamill.

In the session of 1881-2 the Society was reorganized as The Toronto School of Medicine Medical Society, the Faculty were represented, and steps taken to establish a reading room. The members enrolled were: Aikins, Bell, J. F., Carroll, J. T., Cochrane, C. E., Cuthbertson, W., Clendenan, G. W., Coulter, R. M., Cleland, G. S., Colliver, M. K., Draper, J. S., Drake, E. P., Dobson, F. J., Duncan, J. T., Elliott, Hutton, J. G., Hewish, E. M., Jakes, W., Went, F. D., Lafferty, J. F., Laws, R., Montague, W. H., McMahon, T. F., McDowell, E. C., Patterson, J. W., Richardson, Ross, J. H., Robinson, W. J., Stewart, S., Wattam, G. S., Wild, J. J. Willoughby, J. H. C., Watson, A. D.

The following officers were elected: President, Dr. Adam Wright; 1st Vice-President; J. T. Duncan, 2nd Vice-President, Cuthbertson; Recording Secretary, F. J. Dolson; Treasury, J. W. Patterson; Corresponding Secretary, G. W. Clendenan; Curator, J. Stewart; Councillors: Elliott, J. R., Montague, W. H., Aikins, Robinson, W. J., Richardson.

During this session Mr. E. A. Smith of the City Pharmacy offered for competition among the members two prizes, first a pocket medicine case for the best paper upon the therapeutics of opium, mercury and belladonna, and a certified clinical thermometer for best essay on the use of the thermometer in the treatment and diagnosis of disease. The judges were to be Drs. A. McPhedran, J. H. Richardson and Adam H. Wright. Several pages are devoted to very serious discussions regarding these prizes and the conditions of their award, but apparently no theses were ever presented.

The inaugural address of the President, Dr. A. H. Wright, was given January 14, followed by a discussion of a paper read by Mr. W. H. Montague on "The Causes of the present epidemic of Typhoid Fever," in which

Dr. J. E. Graham, C. O'Reilly, I. H. Cameron, G. B. Smith, Jos. Workman and J. H. Richardson took part.

"On behalf of the General Committee Mr. S. Stewart reported the papers on file in the Reading Room, Globe, Mail, World, Grip, Varsity, Acta Victoriana, Canadian Illustrated, Canadian Monthly, all at a cost of about \$10.50."

Two hundred copies of the By-laws were ordered printed and the motto, "Adversus major per secundus," to appear thereon.

Among the names proposed for membership is recorded that of Miss Gussie Stowe, for whom a ballot was demanded. The result was 16 yeas, 11 nays; a three-quarter vote was necessary and the President declared Miss Stowe not admitted. It was moved by Mr. Patterson, seconded by Mr. Cleland that although the Society has declined to receive the name of Miss Stowe as one of its members, yet as the Faculty of this School has granted Miss Stowe all the privileges of a student of the School, that an invitation to the use of the Reading Room and attendance at all regular meetings be extended to Miss Stowe and that the Corresponding Secretary be instructed to give the necessary notice of this resolution.

In amendment—moved by Mr. McMahon, seconded by Mr. Drake, that the words 'and attendance at all regular meetings' be struck out. The motion as amended was carried.

For the next session the Canadian Monthly, Graphic and London Lancet were added to the Magazines on file.

The papers read during the year were "Bacteria and Their Relation to Disease," by S. Stewart, B.A., "Science Falsely So-called," by Dr. J. H. Richardson, with Dr. Jos. Workman in discussion. Three honorary members were elected: Dr. Joseph Workman, Dr. Daniel Clarke and Prof. Wm. Osler.

The final report showed in the Reading Room ten medical journals, one journal of general science, two religious journals, three college journals, three illustrated, and fifteen newspaper-

ers. New books to cost \$150.00 were promised with numerous gifts from the Faculty, "with every prospect of additions being made from time to time until it is second to none on the continent!"

During the session 1882-3 the following papers were read and discussed:

Dr. J. Ferguson—Diseases of the Throat.

Mr. Hearn—Hydropathy.

Mr. Carlton—Union of the Toronto Schools of Medicine, discussed by Drs. Workman, Cameron, A. H. Wright and Mr. Bourke.

Dr. Ferguson—Venereal Diseases.

Mr. S. Stewart—Two Cases of Ulcer.

Dr. Duncan—Simulated Rheumatism.

Dr. J. S. King—Venereal Diseases.

Mr. C. M. Foster—Vivisection.

There were 102 members, 9 meetings held, average attendance 25. The officers were A. H. Wright, President; J. A. Meldrum and J. W. Patterson, Vice-Presidents; James Spence, Secretary, with A. F. McKenzie pro tem; H. H. Wright, Curator.

For 1883-4 Dr. J. E. Graham was President, S. Stewart and Mr. Broadfoot Vice-Presidents, Harry S. Martin Secretary, G. A. Peters Treasurer. Dr. Graham's inaugural address was upon The History of Medical Education in Toronto, with an account of the establishment of the Toronto School of Medicine and Trinity Medical School.

Dr. Reeve offered two prizes of ten dollars each, (1) for the best dissection of the sympathetic nervous and lymphatic system of the thoracic and abdominal cavities, (2) for the best thesis on the lymphatic system and its diseases.

C. M. Foster read a paper on Locomotor Ataxia, H. S. Martin on Suprapubic Lithotomy, Mr. Bascom on Dyspepsia, Professor Montgomery on the place biology should occupy in the medical curriculum. Pathological specimens were usually demonstrated.

Much space is devoted to the growing Library and \$70.00 was voted for

books. Dr. Graham gave \$25.00, and there were many gifts from the Faculty. Total expenditures for books, journals, etc., was \$239.00.

On December 14, 1883, nine months after Koch had demonstrated the etiology of tuberculosis, there was a debate on the subject, "Resolved, that Phthisis is contagious," opened by Dr. Leeming Carr for the affirmative. Mr. A. F. McKenzie followed in the negative, James Spence continued the argument for the affirmative and was followed by Mr. C. M. Foster for the negative. In spite of Koch's demonstration it would appear that Mr. C. M. Foster and Mr. A. F. McKenzie presented their arguments the more forcibly, for by a majority vote the decision was given in favor of the negative!

Following the debate a by-law was passed limiting each speaker in future debates to fifteen minutes!

In the minutes of January 25, 1884, this appears, "Moved by D. D. Ellis and seconded by Mr. Bascom and resolved, that as far as the students of The Toronto School of Medicine are concerned the statements published in the World of January 24th are false in every particular, as there is not the slightest dissatisfaction among the students regarding the management of either the School or the Toronto General Hospital, and that a copy of this resolution be sent to the Toronto World."

For the session 1884-5 Dr. W. H. B. Aikins was President and L. L. Hooper Secretary. The papers read were:

Diphtheria—W. J. Greig.

Jaundice—Mr. Howells.

Hip Joint Disease—Mr. L. Carr.

Chorea—Dr. H. Martin.

Medical Ethics—Dr. W. W. Ogden.

Homeopathy—Dr. J. H. Richardson.

Examination of the body in suspected poisoning—W. J. Mustard, B.A.

Notice was taken of the death of Dr. Frank J. Dolson, a former Secretary. The Library had grown to 144 volumes and 24 medical journals were received.

In 1885-6 W. H. B. Aikins was again President, with J. W. Mustard and John Leeming Vice-Presidents, George H. Shaver Secretary.

Papers and addresses: Dr. L. McFarlane, The Etiology of Hip Joint Diseases; Dr. A. H. Wright, Impotence; Dr. R. A. Reeve, The Therapeutics of Applied Electricity; Mr. Mustard, Mercury and Its Therapeutic Action; Mr. Weld, Experiences in the North-West with the Red Cross Corps; Mr. Rae, Experiences in the Saskatoon Hospital.

The elections for the next year were held at the final meeting in March. "Great interest was taken in the meeting, the attendance being exceptionally large. After the election the retiring President, Dr. W. H. B. Aikens, entertained the members of the retiring committee at Thomas' Chop House, King street west. The sumptuous spread which had been provided by the worthy Doctor received marked attention, and the remainder of the evening was spent in conversation, toasts, speeches, etc. Altogether a most enjoyable time was passed, all with one consent voting the doctor a right jolly good fellow."

For the session 1886-7 the officers included Dr. L. McFarlane, President; J. Vrooman and C. Langford Vice-Presidents; A. M. McFaul and F. W. Kitchen, Secretaries; A. H. Halliday, Curator.

The speakers were J. H. Richardson, Mesmerism or Animal Magnetism; J. Ferguson, Pelvic Haematocoele; Mr. Acheson, B.A., The Histology of Inflammation; A. T. Carson, who kept the audience in roars of laughter with an exhibition of ventriloquism; G. A. Peters on Examination of the Urine; Mr. Acheson on the Inaccuracies of some of the Chemical Tests; Dr. Sweatnam, Bismuth Sub-Iodide in the Treatment of Wounds; W. H. B. Aikins, Bacteriology; J. Ferguson, Magnetism a False Cure.

With the opening of the session 1887-8 the Society becomes the Univer-

sity of Toronto Medical Society. Dr. McFarlane is again President. Mr. E. T. Manes and D. McKay Vice-Presidents, and W. Egbert Secretary. The first speaker to appear before the Society now in the University, was Professor Ramsay Wright, whose subject was "Recent Researches in Cerebral Localization from a Pathological Standpoint," using diagrams "thrown on a canvas by a Magic Lantern." December 2, 1887, "The announcement that Dr. Ferguson would give an address was sufficient to bring out a large number of students who greeted the speaker with prolonged cheers" He spoke on Homeopathy. Other addresses during the session were given by Daniel Clarke and Ramsay Wright. Mr. F. S. Bennett, Mr. J. S. Fothergill and Mr. Hamilton appear to have been the popular entertainers during the session. At the close of the first session in the University the following officers were elected for the ensuing year:

President—Adam H. Wright.

1st Vice-President—Dr. H. A. Yeomans.

2nd Vice-President—Mr. G. A. Shannon.

Cor. Secy.—T. S. Webster.

Rec. Secy.—W. C. Morrison.

Treasurer—J. R. Arthur.

Curator—J. C. Smith.

Councillors—T. Sandison, C. F. McGillivray, T. E. Bennett, H. A. McColl.

This is a summary from the minutes as recorded by the Secretaries for the first ten years of the Society, during which time it was successively The Toronto School of Medicine Literary and Debating Society, the Toronto School of Medicine Medical Society, and finally The University of Toronto Medical Society. Some of the records are scant, in other years the reports of officers and committees are given in full.

Should this summary prove of interest, the succeeding ten years' records may be abstracted for the readers of this journal.

Bright's Disease.

III. DIFFERENTIAL DIAGNOSIS, PROGNOSIS AND TREATMENT.

By F. S. BRIEN, B.A., '33; W. J. HENDRY, B.A., '33; R. B. KERR, B.A., '33.

DIFFERENTIAL DIAGNOSIS.

With the complete history, symptomatology, and clinical manifestations of a given case at hand, the conditions most likely to give rise to confusion in the diagnosis of Bright's Disease are, in the main, suppurative lesions of the urinary tract, and cardiovascular disease.

Pyelitis, Pyelonephritis, Pyonephrosis: These may be differentiated from acute nephritis by the history of chills and fever, and the presence of large numbers of pus cells in the urine.

Cardiac decompensation: This condition is commonly confused with sub-chronic nephritis. However, a carefully taken history will point to a cardiac lesion in the one case and a renal lesion in the other. Among others, the following physical findings indicate a cardiac lesion—murmurs, thrills, arrhythmia, dilatation of heart, diffuse weak apex beat, heart sounds indicative of a poor muscle, enlarged liver, and moisture in the lungs. The presence of exudates in the retina indicates a renal lesion; retinal haemorrhage may occur in either cardiac or renal lesion. The response of cardiac oedema to diuretics is more marked than that of oedema of renal origin.

Arteriosclerosis: Many cases of arteriosclerosis are confused with chronic nephritis, since both may show albumin and casts in the urine, urinary frequency, high blood pressure, and cardiac enlargement. In arteriosclerosis the night frequency only occurs with excessive fluid intake. Kidney function tests show little impairment in about 85% of cases of arteriosclerosis. The remaining 15% show impairment accompanied by inflammatory changes (malignant hyperten-

sion). The systolic blood pressure in arteriosclerosis is usually above 200 mm. Hg., which rarely occurs in uncomplicated nephritis. The fundus manifestations are most useful in differentiating chronic nephritis, malignant hypertension, and "pure" arteriosclerosis.

The retinal changes in chronic nephritis and malignant hypertension have been described in the symptomatology of these conditions.

In simple arteriosclerosis the fundus shows the following picture. The size of the veins is unaltered; that of the arteries is not consistently changed. When intimal thickening occurs, it is usually patchy in distribution. This is shown by contractions in the blood column attending with regions where its width is normal. Usually the light streak is about one-fourth the width of the artery. In medial sclerosis this becomes wider and takes on a burnished copper appearance. Sometimes it appears to be finely beaded. Distinct perivasculitis seldom occurs in the simple arteriosclerotic fundus. Where the vein and artery cross, the vein is often faintly visible through the normal artery wall and is, of course, visible right up to the artery wall. In arteriosclerosis the vein becomes invisible for some distance from the arterial blood column, due to the increased thickness of the arterial wall. The papilla shows slight change in simple arteriosclerosis, apart from changes in the vessels. Haemorrhages may be present in simple arteriosclerosis, particularly if the blood pressure be raised; commonly they are superficial and flame-shaped; round punctuate haemorrhages in the deeper layers of the retina may also occur. Chorio-retinitis is sometimes seen in simple arteriosclerosis (also in syphilis,

tuberculosis and nephritis). Deep to the retinal vessels, pigmentation and depigmentation are found, most frequently in the macular and peripapillary areas.

It must be realized that these features are frequently found in varying degree in the fundi of malignant hypertension and chronic nephritis, as a basis upon which the characteristic findings of these conditions are superimposed.

Myxoedema: Atypical forms of myxoedema, particularly those unaccompanied by marked mental changes, and which show dependent oedema and progressive cardiac weakness, with some rise in blood pressure, may be confused with nephritis, due to the presence of albumin, casts, and possibly blood, in the urine. History, basal metabolic rate, and response to thyroid treatment are differentiating features of this condition.

PROGNOSIS.

Acute Nephritis: 50% of cases of acute nephritis recover completely leaving no residual signs or symptoms. It must be remembered that acute nephritis is not a disease lasting for six to eight weeks, as formerly considered, but one in which treatment should be carried out up to one year after the onset, or as long as improvement is seen.

Subacute Nephritis: This condition presents a much less favourable prognosis. The so-called "stormy" case usually ends fatally in uraemia in from six months to three years.

Subchronic Nephritis: A cure is out of the question. The cases formerly placed in this class and subsequently considered cured, may be looked upon as inadequately treated cases of acute nephritis. The situation is not hopeless, however, for the progress toward renal failure may be exceedingly slow and the patient may live for many years. The cause of early death in this phase is due to repeated exacerbations.

Intercurrent diseases play a part in the prognosis, rendering it less favourable.

Chronic Nephritis: These cases come to a fatal termination in a few months or at most in a few years, and may be hastened to the exitus by an acute exacerbation. Amongst the findings helpful in prognosis are—the level of fixation of specific gravity (the lower the level, the worse the prognosis), increase in N.P.N., blood creatinin, and blood indican. Increase in N.P.N. and creatinin marks a progressively poorer prognosis. A creatinin value of over 5 mgms. per 100 cc. indicates a fatal termination within about 4 months. The onset of the retinitis of chronic nephritis or the appearance of the macular star usually signifies death within 2 years.

Malignant Hypertension or Combined Nephritis: The prognosis of this condition is invariably fatal. Death may occur from uraemia, heart failure or cerebral accident. The renal form of the disease progresses rapidly. Over 50% of the cases end fatally within one year of their coming under observation, while few cases live over four years.

TREATMENT.

Nephrosis: The chief aim in the treatment of nephrosis is to place the injured organ in as complete a state of rest as possible. To accomplish this, absolute rest in bed is, of course, an essential. Restriction of mineral salts and fluid is helpful in reducing the work of the kidney. In cases with massive oedema, free saline purgation is of great value. Diuretics such as urea and salyrgan are also useful.

Foci of infection should be investigated. However, it should be remembered that any surgical procedure in an oedematous patient is complicated because of the increased danger of infection.

In nephrosis caused by specific poisons, notably mercury, it is important to carry out gastric lavage and colonic irrigation repeatedly with a 2% solu-

tion of sodium thiosulphate (to precipitate the mercury). Intravenous administration of 20 grains of sodium thiosulphate is also indicated. Since, in this type of nephrosis, oedema is not a marked feature, fluids and soluble carbohydrates may be administered to advantage. If uraemia develops, purgation and powdered charcoal are of value.

Acute Nephritis: Rest in bed is essential. In cases which are seen early in their illness, complete starvation of food and fluid for two or three days frequently results in a marked improvement in the clinical picture. This rigorous treatment may be mitigated by allowing the patient small quantities of fruit juices. However, in cases which do not come under observation until about two weeks after the onset of the disease, it is found that the above complete starvation treatment is not of advantage.

After the initial restriction of food, in the first instance, or as the immediate treatment in the second instance, small amounts of milk, 500-800 ccs. per day are given, other fluids being withheld. Thus one attempts to maintain the intake of food and fluid within the limits of kidney efficiency. Even though the diet contains less than the patient's basal caloric requirement, the resultant undernutrition is of little consequence, provided that vitamin C is supplied as orange or tomato juice. Purgation with salines and hot packs to the lumbar region are of value. In extremely severe cases spinal anaesthesia, stripping the renal capsule and section of the renal nerves have been suggested.

Urine examinations are made daily to determine the tolerance of the patient to his food intake. When the urine has been albumin-free for a week, the daily quantity of milk is increased by 200 ccs. The patient's diet is maintained at this level until the urine is albumin-free for another week; thus his intake is increased gradually. Foods high in carbohydrate and fat,

but low in protein, are added as the patient improves. Salt is restricted due to its tendency to produce oedema. Vegetables are limited because of their water-carrying power and inorganic salt content. Ideally, the diet should be increased only at a rate which parallels the ability of the kidney to excrete its waste products. When an adequate caloric intake is reached the patient may be allowed up. This means a sudden increase of several hundred calories and a considerable increase in the breakdown of proteins resulting in a period of stress in so far as the kidney is concerned. Hence careful observation is essential. If the clinical and laboratory findings indicate no marked break in kidney tolerance, the protein intake may be stepped up to 60-75 gms. per day. While salt may be permitted in the cooking of the patient's food, it should be avoided at table for one year after clinical cure has been attained, during which time also protein intake should be restricted to the above value.

Foci of infection should be investigated and removed when urinary findings are normal and manifestations at the focus are quiescent.

Subacute Nephritis: In the early part of this condition treatment is similar to that of acute nephritis except that readily demonstrated foci of infection should be removed in spite of the activity of the lesion, either in the kidney or in the focus. In the later months treatment is similar to that of chronic nephritis.

Subchronic Nephritis: The outstanding principles in the treatment of a subchronic nephritic are regulation of diet, observation, elimination of foci of infection, and avoidance of any infections or conditions which may lead to an acute exacerbation. Salt, fluid and protein intakes should be restricted, dependent upon the amount of damage to renal efficiency. Daily saline aperients are recommended. Exacerbations are treated as acute nephritis.

Chronic Nephritis: In the chronic

nephritic the obligatory polyuria demands an increased water intake. Protein must be limited and what is taken should be readily assimilable—vegetable protein being on the whole less satisfactory than animal protein. Mineral salts should be limited. Attention to foci of infection is indicated as before. Morning salines are advised. Symptomatic treatment is indicated for headache, and insomnia. When an exacerbation occurs, treatment is similar to that of acute nephritis, except

that no restriction of fluids is advisable. Intravenous glucose, up to 3000-4000 cc. may be necessary.

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Facial Nerve Paralysis.

By H. H. HYLAND, M.D., M.R.C.P.
 Department of Medicine.

The facial nerve is the most commonly paralyzed nerve in the body. At first glance the anatomy of the nerve and its supranuclear connections may seem rather involved, but, if certain fundamental facts are kept in mind, little difficulty should be experienced in localizing the position of a lesion of this nerve. In the following account an attempt will be made to describe briefly the physical signs of lesions in various parts of the course of the facial nerve. Mention will be made of the commoner causes of the lesions and certain anatomical facts will be emphasized. For purposes of description, lesions at different levels may be classified as peripheral, pontine and supranuclear.

Lesions of the Peripheral Nerve.

Anatomical Considerations: The motor root emerges from the lateral aspect of the pons in the cerebellopontine angle where it is separated from the acoustic nerve by the sensory root of the facial nerve (pars intermedia). In company with the acoustic nerve the motor and sensory roots pass through the internal auditory meatus. At the bottom of the meatus the facial nerve enters the fa-

cial canal, passes between the cochlea and vestibule, and turns downwards behind the tympanic cavity to emerge from the skull by the stylomastoid foramen. During its course in the facial canal it gives a branch to the stapedius muscle. On leaving the skull it runs deep in the substance of the parotid gland and divides into its branches behind the ramus of the mandible. The branches supply the superficial muscles of the head, face and upper part of the neck with the exception of the levator palpebrae superioris, which is supplied by the oculomotor nerve.

The sensory fibres convey impulses of taste from the anterior two-thirds of the tongue by the chorda tympani. The latter travels with the lingual nerve for a distance, then crosses over to join the facial just before it emerges from the stylomastoid foramen. Superficial sensation from the internal auditory meatus, the tympanum and the auricle, together with deep muscle and tendon sensation from the face, are also conveyed by sensory fibres in the facial nerve. The sensory fibres pass to the geniculate ganglion in the facial canal and from thence by the sensory root to the brain stem nuclei. The sensory

root leaves the motor root at the internal auditory meatus.

Causes of Peripheral Lesions:

1. Tumours in cerebellopontine angle (viz.: acoustic neuromas and tumours of cerebellar hemispheres).
2. Basilar meningitis of all types (particularly syphilitic and tuberculous meningitis).
3. Bell's palsy (probably the result of periostitis in region of stylo-mastoid foramen).
4. Middle ear diseases (by extension to facial canal in petrous portion of temporal bone).
5. Aneurysm of the external carotid artery (involving the nerve as it crosses the artery before dividing into its terminal branches).
6. Direct trauma (viz.: birth injury).
7. Parotid gland tumours (viz.: mumps).
8. Polyneuritis (the facial is affected together with other nerves).

Clinical Signs and their Interpretation.

Depending on the severity of the lesion, there will be paresis or paralysis of all the muscles supplied by the nerve on the affected side. At rest that side of the face is flattened out and the nasolabial fold is less marked. The corner of the mouth is dropped, and the palpebral aperture is widened. Blinking is impaired or absent on the affected side. On voluntary or emotional movement the asymmetry of the face is accentuated, e.g., on showing teeth, smiling, whistling, blowing out cheeks, frowning, wrinkling forehead, and attempting to close the eyes tight. When attempting these movements the weakness of the various muscles involved is very obvious by contrast with the good response on the normal side of the face. Owing to paralysis of the orbicularis oculis, the corneal reflex may not be elicited on the affected side although

corneal sensation is intact (trigeminal nerve). In facial paralysis, however, the consensual corneal reflex is present and there is blinking of the healthy eye on testing the affected side, whereas in trigeminal involvement there is no consensual reflex.

If the lesion has attacked the nerve within the facial canal certain findings are likely to be present. Taste will be lost on the anterior two-thirds of the tongue due to involvement of the chorda tympani. Hyperacusis is sometimes present, resulting from paralysis of the stapedius muscle. Pain about or within the ear is common in lesions in this situation owing to involvement of the sensory fibres. Lesions affecting the nerve after its emergence from the skull are not associated with loss of taste. Lesions occurring in the cerebellopontine angle are accompanied by signs of acoustic nerve involvement and commonly show evidence of disease of neighbouring structures, e.g., cerebellum, pyramidal tracts, abducens and trigeminal nerves, etc.

About ten days after the onset of peripheral facial nerve paralysis the electrical reactions show changes in the direction of reaction of degeneration. Subsequently the muscles may develop considerable atrophy but never fibrillation. Contractures are liable to occur. Herpes, in the region of the external auditory meatus and the ear drum, may occur with certain lesions of the geniculate ganglion, and may be associated with facial paralysis.

Pontine Lesions.

Anatomical Considerations: The motor fibres originate in the nucleus in the lower part of the pons. The fibres run backwards, looping around the abducens nucleus and then downwards and forwards to emerge from the pons between the olive and inferior cerebellar peduncle. Many important nuclei and tracts lie in close proximity in this location, with the result that lesions commonly give rise to characteristic syn-

dromes. Lesions within the pons may involve the nucleus, the nerve in its course through the pons, or both.

Causes of Pontine Lesions.

1. Progressive bulbar palsy (viz.: progressive muscular atrophy).
2. Polioencephalitis (brain stem type).
3. Encephalitis lethargica.
4. Diphtheria; rabies; tetanus.
5. Syringobulbia.
6. Vascular lesions of the brain stem (thrombosis, haemorrhage, and embolism).
7. Tumours (viz.: gliomata and tuberculomata).

Clinical signs and their interpretation. The appearance of the facial paralysis at rest and on voluntary or emotional movement is similar to that already described. In certain slow progressive lesions affecting the nucleus (P.M.A.) the muscles may waste one at a time. This never happens in peripheral lesions, but is possible in nuclear lesions because the arrangement of the ganglion cells corresponds to the muscles they supply. Taste will not be affected in a pontine lesion because the sensory fibres conveying taste terminate in a nucleus in the medulla continuous with the glossopharyngeal nucleus. Hyperacusis will not occur, nor will there be pain in the region of the ear. In slowly progressive nuclear lesions fibrillation may be seen in the affected muscles (viz.: progressive bulbar palsy and syringobulbia).

With a unilateral lesion, the common finding is paralysis of the facial and probably the abducens nerve on the side of the lesion, with signs of pyramidal tract disease and sensory impairment in the limbs of the opposite side—these tracts having crossed in the medulla and spinal cord.

The abducens nerve is the commonest cranial nerve to be involved with the facial in pontine lesions, but

the oculomotor, trigeminal, acoustic and vagus are frequently affected also.

Supranuclear Lesions.

Anatomical considerations. The upper motor neurone fibres of the facial nerve pass from the frontal cortex through the anterior part of the internal capsule to the pons by way of the corticobulbar tract which occupies the mesial part of the peduncles. The fibres cross in the midline in the upper part of the pons so that the facial nucleus of one side is controlled by the opposite frontal cortex. The muscles of the upper part of the face, particularly the frontalis muscles, appear to have bilateral representation in the cortex.

The supranuclear fibres for emotional expression (laughing and crying) travel by a separate path from the above via the thalamus—the so-called cortico-thalamo-mesencephalic path which terminates in the facio-respiratory nuclei and is only partially under pyramidal inhibitory control.

Causes of Supranuclear Lesions.

1. Cerebral haemorrhage, thrombosis or embolism.
2. Encephalitis of all types, including abscess.
3. Meningovascular syphilis.
4. Disseminated sclerosis.
5. Amyotrophic lateral sclerosis.

Clinical signs and their interpretation. The paralysis resulting from a supranuclear lesion differs materially from that already described and should never be confused with it. The lower part of the face is principally affected; the orbicularis oculi is often weak but never completely paralyzed; and the frontalis often escapes entirely. The appearance of the lower half of the face at rest and on voluntary movement is similar to that found in lower motor neurone paralysis, but it differs in that no evidence of paralysis is detected on emotional movement, whereas, in lower

motor neurone lesions, voluntary movement and emotional movement are equally affected. In supranuclear lesions taste is not affected and there will be no hyperacusis. The electrical reactions are not altered and there is no pain, atrophy or fibrillation.

It is often valuable to distinguish between supranuclear lesions which are cortical in origin and those which occur lower down. If the facial paralysis is associated with a hemiplegia on the same side, the lesion is usually subcortical—probably in the internal capsule. In cortical lesions the face may be paralyzed alone, or possibly the arm on the same side may be involved along with the face. Jacksonian fits occurring in the paralyzed side of the face indicate cortical involvement.

Thalamic Lesions. Occasionally lesions in this situation cause a paralysis of the face on the opposite side, which is only apparent on emotional move-

ment (smiling, laughing or crying). No paralysis is observed at rest or on voluntary movement. This is due to the lesion involving the cortico-thalamo-mesencephalic path for emotional expression, the cortico-bulbar fibres having escaped.

Bilateral Facial Paralysis. This may occur with lesions in the peripheral nerves, e.g., polyneuritis; in pons, e.g., tumour; and in the supranuclear fibres, e.g., associated with bilateral hemiplegia, so-called "pseudobulbar palsy." The clinical signs are similar to those described for unilateral paralysis.

Certain diseases, primary in the muscles, may produce an appearance simulating bilateral facial nerve paralysis. This is seen in myasthenia gravis and the facio-scapulo-humeral type of muscular dystrophy. These conditions will be readily recognized by the history, associated findings and the electrical reactions.

The Treatment of Acute Appendicitis.

By I. G. MACDONALD, M.D., C.M.

Department of Surgery.

Following upon the introduction of general anaesthesia and asepsis, a full generation ago, abdominal surgery became a comparatively secure and common pursuit for the general surgeon. Coincident with this, immediate laparotomy and appendectomy became the standard treatment for all those cases hitherto diagnosed as "intestinal inflammation," following the pioneer work of Murphy of Chicago. During these early years there appeared a large volume of literature on the subject of acute appendicitis, as valuable clinical, operative and pathological findings were added. With this came in later years improvements in operative technique, as the McBurney and Battle types of procedures. Ultimately, the clinical dictum, still adhered to by many

surgeons of repute, held sway—"when the diagnosis of acute appendicitis has been established, operation must be resorted to without delay."

This remains, in the majority of cases, the best standard of treatment. But there are certain exceptions to this excellent rule, and these are dependent upon the accuracy of the conclusions one draws from physical examination. Such conclusions should result in a definite picture, in general, as to the condition of the appendix and its surroundings. One can expect to remain in some doubt as to the details of the exact pathologic process present in the appendix, but still be clear upon certain facts recognizable with ease, and of utmost importance in treatment. It must be recognized first of all that there

are many cases presenting the entire symptomatology of acute appendicitis which do not subsequently show any gross or microscopic lesions. These are well termed "appendicular colics," due practically always to faecaliths within the appendix. Absence of fever and leucocytosis are most typical of this class, and appendectomy the safer course to pursue. In the following it is proposed to consider only such cases as rest upon an actual inflammatory lesion. There are thus four questions which must be considered in every case diagnosed as acute appendicitis:

1. Is the inflammation limited to the appendix and its immediate neighborhood?

This condition represents what is usually termed as the early stage, for in most instances it is only when the inflammatory process has spread from the appendix proper to the nearby serous surface that the clinical signs of an "attack" appear. The peritoneum is slightly reddened, some fibrin may be present on the appendix, and the abdominal cavity may contain some early exudation. Such an exudate may be turbid and full of leucocytes, but is practically always sterile. This early stage may last more than one or two days. The patient must be considered in this stage if the upper abdomen and the left lower quadrant are still soft and not tender, if there is no pain in the left lumbar region, and if pain on pressure and rigidity or resistance are limited to the suspected

site of the appendix, without any extensive dullness or definite resistance. With these criteria fulfilled, there is no other choice than immediate operation and appendectomy. Should free fluid be present, it is advisable to take a culture if facilities be available. The finding of even large amounts of turbid or opalescent fluid in the presence of a non-perforated appendix should never prevent one from closing the abdomen without drainage. Drainage is almost certain to result in wound infection and thus lengthen the number of hospital days.

2. Is an inflammation which has gone beyond the first stage localized or generalized?

In this event, the process is no longer appendicular and peri-appendicular with early aseptic exudation, and it must be determined again by physical examination how far the inflammation has travelled. If the abdomen is everywhere sensitive to pressure, with tenderness in the flanks, and if there is extensive muscular resistance amounting to rigidity across the lower abdomen, then there is obviously widespread peritoneal involvement. If it is the patient's unfortunate lot to be seen in this condition, it is the surgeon's duty to propound to himself the third question:

3. Upon what does this general involvement of the peritoneum depend?

Two very different conditions may be responsible and the treatment may

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of necessity differ. Most favourable are those in which the signs mentioned merely indicate a marked generalized early exudation, which will in all probability subside after the inflammatory area has been encapsulated, should they go untreated. These are conditions which occur when the attack is at its commencement, with pulse and temperature in correct relation to each other. The muscular rigidity is neither severe nor extensive, the vomiting has ceased, and the patient does not appear septic. In these cases, again, immediate appendectomy and closure without drainage is the only procedure of wisdom.

Most grave of all cases of appendicitis are those where such a general infection of the peritoneum is a severe septic type, due to one of two complications:

- (a) Acute gangrene of the appendix.
- (b) Perforation and flooding of the peritoneal cavity with septic material.

The exudate contains more bacteria than leucocytes, in contradistinction to the early aseptic exudate. It is in such cases as these that the controversy centres as to the wisdom of "delayed treatment." The question is still far from settled, and will remain so until comparative statistics from large surgical services have become valuable by reason of a greater number of cases treated. At present, it may be said that where the fully developed picture of general peritonitis is present, and the patient obviously septic, probably in the third or fourth day of illness, it is the course of wisdom to refrain from opening the abdomen at least until the patient's condition has been improved by other treatment. Suitable measures are continuous drip intravenous with the patient in high Fowler's position, heat to the abdomen, small enemata only for distension, suitable stimulants hypodermically and nothing by mouth. Most cases which show such unfavorable signs as pallor, cyanosis,

rapid thread-like pulse, subnormal axillary temperature with a high rectal temperature, and meteorism may be treated on this plan. If marked improvement occurs without evidence of abscess formation, operation may then be undertaken. Should a localized mass develop and the patient continue to improve, then expectant treatment may be continued as described below. There are occasions when it is possible to say with fair accuracy from the history that a perforation has occurred at a definite time. If the patient is seen two to four hours after such an accident, immediate operation should be undertaken, and appendectomy performed and provision made for liberal drainage.

There are, of course, many gradations of sero-purulent peritonitis intermediate between the two extremes just described, and there are many cases in which it is most difficult to determine whether a spreading peritonitis is due to actual perforation, or evidence of a highly virulent infection. In all such cases immediate operation should be resorted to and, if necessary, a drainage tube passed to the pelvis.

In the event of having decided that the extension of inflammation is not generalized, but become localized, the fourth question very naturally presents itself as:

4. What is the position of such a localized mass and has it gone on to abscess formation?

The outlines of an abscess must be determined by palpation and percussion. Even more important is to determine whether the abscess is extra-peritoneal. Pronounced rigidity of the anterior abdominal wall in the right lower quadrant, filling of the pelvic fossa as determined by rectal examination, indicate an intra-peritoneal position, which will be made certain if it projects into the abdominal cavity. Extra-peritoneal abscess is shown by the appearance of a phlegmon in the lumbar region, or contraction of the flexors of the hip.

Tracking of the abscess under Poupart's ligament indicates a sub-fascial position i.e., under the fascia of the iliacus muscle. If rectal symptoms appear, as diarrhoea and mucus, the abscess is situated in the pouch of Douglas.

Acute appendicitis which has gone on to the formation of a mass is one condition where the scalpel should be carefully left on the shelf, preferably under lock and key, until such time as one of three things has come to pass. First, and most happily, the mass has resolved, the patient recovered, and returned to his normal pursuits for a period of three to six months, when an "interval" appendectomy may be performed. Secondly, if there is abscess formation and pointing, it should be opened with a small incision and drained. This should be done with as little anatomical damage as possible, in the right lower quadrant, lumbar region, vagina or rectum according to the location of the abscess. It is thus important to find, by careful local diagnosis, where the smallest incision will suffice. If there is tympany well marked over the right lower quadrant, one should go far out in the loin, and follow inwards re-

tro-peritoneally until the abscess wall is encountered, that the peritoneal cavity may not be flooded with pus. Thirdly, if at the end of a week the general and local signs and symptoms show no signs of subsiding, the presumption is that formation of pus has occurred in the mass, and the abscess should be drained under the same plan as outlined above. Removal of the appendix under such conditions, however, should amount to malpractise.

Summary.

1. All cases of acute appendicitis without perforation should be operated upon immediately, and appendectomy performed without drainage.

2. Every case seen within a few hours following perforation should be subjected to appendectomy, with or without drainage.

3. Some controversy exists as to the wisdom of immediate operation in severe, generalized peritonitis following perforation. For all practical purposes it may be said that if the patient's condition is such that the shock of operative procedure will be borne poorly, then delayed treatment may be instituted. Otherwise, operation should be undertaken.

4. Those cases in which a mass is formed should be treated expectantly, and the abscess drained if such develops.

Intestinal Intoxication in Infants.

By A. H. SELLERS, B.A., '32.

Intestinal Intoxication in infants is the name applied to a symptom complex whose cardinal features are diarrhoea, vomiting and drowsiness. It is one of the most interesting and perplexing problems in medicine, and hundreds of infant deaths are due to it each year — in spite of treatment.

It was Finkelstein who, observing that the picture presented by the condition resembled that of an acute poisoning, gave it the name Alimentary Intoxication — a clinically descriptive term which probably should be discarded. Intestinal Intoxication is also

synonymous with the Anhydremic Intoxication of Marriott, Catarrhal Enteritis, Infectious Diarrhoea, Summer Diarrhoea, Diarrhoea and Enteritis and Cholera Infantum.

Rush, in 1773, was the first to describe the condition when he wrote: "An inquiry into the cause and cure of Cholera Infantum" — but it was really not until the middle of the 19th century that the disease was described or definitely recognized in Great Britain. Rush, in his original description, thought that the disease was peculiar to America. However, it has been re-

ported in both tropical and temperate zones on this continent, in Europe and elsewhere.

Incidence.

Intestinal Intoxication in infants occurs both sporadically and in epidemic form, the epidemic months being August, September, and the early part of October. The disease is one of infancy and early childhood — being most frequently seen in the first year of life. In many of the cases there is a history of previous gastro-intestinal disturbance, but this is not constant, and many infants who were previously quite healthy contract the disease.

The infants affected are, on the whole, below standard, and since about 90% of cases occur in bottle fed infants, artificial feeding is evidently a factor of predisposition at least.

Infants from the poorer homes — infants who are improperly fed or who are exposed to unhealthy, unhygienic surroundings, provide most of the cases. It is relatively uncommon in a private practice or in the better-class home. The sexes are equally affected.

Onset and Course.

Both the onset and the course of the disease is very inconstant. The onset may be gradual or sudden. It may be gradual and be preceded by a nutritional disturbance, for example, such as an atrophy or decomposition.

History of a previous upper respiratory infection is usually uncommon. On the other hand, the onset may be sudden and in a previously healthy child. Frequently the history is obtained of loss of appetite followed by a little vomiting and later by diarrhoea. In other cases, diarrhoea may commence suddenly and be the first symptom — the vomiting following later. Diarrhoea alone or vomiting alone is sometimes present — but intoxication without diarrhoea is rare.

The vomiting varies greatly in frequency from 2 or 3 to a dozen or more times a day, and may be very forcible in character — first of food and later mucous and bile.

The stools are loose and watery and yellow to yellowish green or chyle-like in colour. Blood and pus are often observed, and would probably be found in all cases at some stage of the disease — were a careful and diligent search made.

Depending on the dehydration — diarrhoea and vomiting are followed by drowsiness. This frequently begins in 24—48 hours from the onset of symptoms. At first drowsy and uninterested in his surroundings, the infant becomes quite lethargic. Drowsiness varies a great deal even in the same baby — and observations must be made without disturbing the infant, because when aroused the infant may appear quite bright.

At any time, the severe stage may appear suddenly — particularly in premature or atrophic infants — though gradual development of the coma is the rule.

Fever also is variable, and its course is determined by the reaction of the

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patient. It may be slight or moderate, or it may rise suddenly to 104° or even to 106° . At the approach of death, hyperpyrexia or subnormal temperature may occur — the latter predominating in the premature or atrophic infants.

The infant loses a great deal of weight. The eyes are sunken, the fontanelle depressed, the skin dry and inelastic, showing the peculiar pallor of wet ashes and hanging in loose folds. The hands and feet are cold and moist and perhaps cyanosed. The infant presents a mask-like stare, and frequently assumes what Finkelstein described as the "boxer's position."

The respirations, which become deep and rapid, must be observed, and suggest diabetic or uremic coma. The lips become deep pink or red in color and the conjunctiva congested. The mouth frequently remains open. The child, when aroused, often gives a shrill and piercing cry.

The whole picture, then, is dominated by two factors — the dehydration resulting from the vomiting and diarrhoea and the acidosis due to loss of basic substances by the bowel and production of ketone bodies due to starvation.

The blood shows a polymorphonuclear leucocytosis. It is thick and shows raised serum proteins. The capillary blood flow is diminished and the N.P.N. is elevated.

Anuria may occur in some cases — but the urine in others shows a varying amount of albumen — blood cells and casts — pointing to a kidney injury.

The course of the disease in the severe type — including the original cholera infantum — may be very rapid, and some infants die within 24-48 hours of the onset of drowsiness — treatment notwithstanding. These infants show a rapid weight loss, acute desiccation, severe prostration and disturbed consciousness — together with leucocytosis and high fever. These severe cases may follow a preliminary mild attack, especially if improperly or inadequately treated. Vomiting is

early and persistent, and meteorism may be marked. Recovery in these cases may occur over a course of 3-4 weeks.

Moderately severe cases continue to vomit or are drowsy for several days before they seem to get worse or better. The mild type are those most responsive to treatment — vomiting and drowsiness disappearing within 24-36 hours and the infants gradually returning to normal in 6-10 days. These cases show little or perhaps no fever.

Mortality in the severe type is very high — certainly over 50%, and appears to be independent of previous nutritional history of the infant — and indeed, in many cases, the fat, overweight baby shows the least resistance to the disease.

Etiology.

This has been the battleground of Paediatricians for years. Many theories have been advanced and can be classified thus:

1. Due to food. (Czerny, Keller, Finkelstein, Meyer, etc.).
2. Formation of toxic substances by decomposition.
3. Abnormal absorption of catabolites from the bowel.
4. Parenteral infection (mastoid theory). Marriott, etc.
5. Enteral infection.

The earlier investigators were convinced that food was the etiological factor. Finkelstein and Meyer thought, from clinical experiment and observation, that an excess sugar or whey salts could produce the intoxication — being allowed to reach the tissues unchanged — by virtue of an increased permeability of the intestinal wall. Czerny and Keller attempted to show that the intoxication was due to spoiled milk. Moros believed a food disturbance to be the prime factor, with acidosis and dehydration as late manifestations.

The beneficial results of exsanguination transfusion seemed to support the idea of a toxin. It was thought at

this time that this toxin plus the increased permeability were factors.

Infection as the etiological agent has, however, proven itself in recent years, and it is now well recognized that severe intoxications in infants is due not to food poisoning or circulating toxin, but to toxemia of infectious origin.

The evidence at hand, as the result of the extensive work under Dr. Alan Brown at the Hospital for Sick Children, Toronto, strongly substantiates the conclusion that intestinal intoxication in infants is an enteral infection with an organism or organisms of the paratyphoid dysentery group.

Diagnosis.

The diagnosis of the condition is made on the typical symptoms and signs. Diarrhoea, vomiting and drowsiness—fever, dehydration, toxicity of the patient and acidosis. In advanced cases, the diagnosis is suggested at a glance, and the experienced clinician recognizes the condition from the toxic appearance of the patient.

The baby's somnolence, expression, pallor, deep rapid respirations and depressed fontanelle are characteristic. Very young infants are often very drowsy early and if awakened they may show no sign of unconsciousness at all—though they may readily lapse into sleep. In an emaciated child with depressed fontanelle and deep rapid respirations the diagnosis is probably intestinal intoxication. Encephalitis and meningitis must be thoroughly ruled out. In meningitis the fontanelle is as a rule protuberant, and general examination and laboratory investigation give other evidence of meningeal involvement.

Prognosis.

In general, prognosis depends on age—the younger the patient the greater is the danger.

The duration of the disease before the institution of treatment and "resistance" of the patient are factors.

If there is improvement in 24-48

hours, recovery is likely. Atrophic babies have an unfavorable outlook. Previous nutritional condition and degree of dessication are factors also. Acidosis, when persistent and present to an extreme degree, may be taken to indicate that water loss has been extreme. Intercurrent infection—nasopharyngitis and otitis media—may render the course more severe, and intercurrent broncho-pneumonia is a real danger.

Pathology.

The most constant pathological finding is that of an enlarged and fatty liver—which is found in 80% of cases. The cut surface shows frequently a yellowish green appearance, and microscopic section reveals a fatty infiltration.

In many of the cases, the gastrointestinal tract in the gross appears normal, though all variations from a mild congestion of the mucous membrane of the lower bowel—to ulceration of the wall occur. In severe cases there is degeneration of the epithelium together with swelling of Peyer's patches. In many cases the mastoid antra have been found to be full of pus. The kidneys show a mild cloudy swelling with mild epithelial degeneration in the convoluted tubules. In a very acute case, dying in 24 hours, the pathology may be negligible, whereas a mild protracted case will show well defined lesions.

In any case, the severity of the clinical symptoms is much out of proportion to the rather insignificant pathology found.

Blood Chemistry.

Some conception of the changes in blood chemistry which occur in these babies is of value in discussing the treatment of the condition.

The normal blood chemistry is modified in intestinal intoxication by two factors:

(1) Dehydration, which is due to loss of water from the body by diarrhoea and vomiting.

(2) A change in the acid-base balance — which may be in the nature of an acidosis or an alkalosis, more usually the former.

(a) If the vomiting is predominant, much chloride ion is lost — greater than the sodium or base lost, and alkalosis occurs.

(b) If the diarrhoea predominates, which is the rule, the base lost by diarrhoea being greater than the acid lost by vomiting, acidosis occurs, and the deep, rapid respirations of the acidosis state are seen.

(c) The infant by this time has lost considerable weight, is taking no food and is starving. Acetoacetic and B-oxybutyric acid are produced and ketosis wipes out more of the alkaline reserve.

(d) Another factor is impairment of renal function and failure of the kidney to excrete acid phosphate — resulting in retention of acids and still further depletion of the bicarbonate reserve and aggravating the acidosis.

Besides these changes, as one would expect, there is an increased viscosity with great reduction in volume flow. This alone seems sufficient explanation for the failure of physiological processes, and ultimate death.

Treatment.

There is no specific treatment for intestinal intoxication, but there are certain fundamental facts which have been sufficiently supported by clinical evidence to justify general acceptance, and it is therefore possible to compile a routine which is very helpful to those who must treat the sick baby.

The diarrhoea and vomiting, with their dehydrating effect and the consequent starvation, are the absolute causes of the acidosis which precedes death. The following must be combated, and are three in number.

1. Dehydration.
2. Acidosis.

3. Toxemia — resulting from dehydration and toxic absorption.

Fluids:—Water loss must be replaced as rapidly as it is safe to do so, and fluids must be administered in a manner in which they will be retained. Water loss is one of the important factors which aggravates the toxemia.

A direct transfusion of whole blood, 15 cc. per pound of body weight, may be given and repeated in 24 hours if indicated. This provides us with a means of combatting the toxicity by supplying fluids, acids and bases in their normal proportions as well as energy and antibodies. If a case is seen early — within 24 hours of its onset, say, blood transfusion seems to be almost specific, and the baby will show great improvement on the following day. If seen later on, however, blood transfusions are less efficacious.

Loss of body substances in dehydration is almost entirely sodium and chloride. Energetic administration of physiological saline is the basal step in an attempt to treat dehydration. Sodium and chloride ion have been lost by the infant usually to a different extent, and ordinarily one could depend on the kidney to deal separately with the ions according to the needs of the plasma. But renal function is impaired in intestinal intoxication, and sodium and chloride tend to be retained in the concentration in which they are given — which merely aggravates any acidosis which is present.

The chief factor causing impairment of renal function seems to be dehydration, and the chief factor needed to restore it, a surplus of water. This is best provided by glucose solution, since the glucose is burned by the body — leaving the water available for kidney function.

Hence parenteral administration of fluid, 2-3rd as 5% glucose and 1-3rd as normal saline, is found to be efficacious. The glucose solution then does a variety of things: (1) stimulates renal function; (2) provides fluids; (3) dilutes toxins; (4) provides sorely needed cal-

ories for the starved patient; (5) it is antiketogenic; (6) intravenously it hastens absorption of subcutaneous saline; (7) protein sparing effect — reduces rate at which protoplasm is being destroyed.

The routes for administration are—subcutaneous, intra-peritoneal and intravenous. Subcutaneously 10 c.c. per pound of body weight may be given and repeated 2-3 times a day, depending on the rate of absorption. The best site for administration is the subcutaneous tissue of the abdomen. These administrations are reduced as the rate of absorption decreases, but are continued as long as there is need for fluid.

Intraperitoneally, glucose solution is avoided, as it tends to produce abdominal distention, but saline may be given — 20 cc. per body pound weight. Intravenously, 15 cc. per lb. of body weight is given into the longitudinal sinus if the anterior fontanelle is open—otherwise the median basilic vein may be utilized. The nasal drip may also be used. Administration of fluid should be continued until the tissues are saturated and no further absorption is taking place from the interstitial injections — or until the infant is able to take fluid by mouth in sufficient quantities. If desiccation is extreme, all four routes may be used.

Food.—As the baby's tolerance for food is practically wiped out, food should be withheld at the commencement of treatment and milk withheld throughout the entire acute stage—as milk feedings aggravate the diarrhoea, vomiting and toxicity. One must not forget, however, that starvation plays a part in the disturbed acid base balance of these infants and every attempt must be made, therefore, to give easily assimilable food early.

When the vomiting has stopped or is infrequent, one may give either one of the solutions: (1) 15% sugar as 7½% glucose and 7½% dextri maltose—with a caloric value of 18 calories per ounce. On this infants may retain

weight for 10 days—if enough can be given. (2) A solution called C.O.G. (1 part 1% sodium citrate, 2 parts orange juice and 3 parts 10% glucose). It has a caloric value of 10 calories to the ounce,—or the same as half strength milk.

These fluids are given cold, in teaspoon doses every 5 minutes, at the rate of 2½ or three ounces per hour.

If vomiting recurs—gastric lavage with bicarbonate solutions, 1 teaspoonful to the pint, may be done—100-200 c.c. of the solution are left in the stomach.

When the infant has been free from toxicity for 24-48 hours and if vomiting is absent—milk feedings are begun and gradually increased, gradually replacing the fluids described. This period in the treatment is of vital importance, and improper conduct may result in a relapse. It is one in which the infant's tolerance for food is gradually being coaxed along. It is the period of quality gain or repair period.

The citrate in the C.O.G. may first be replaced with barley flour—1 ounce. And later lactic acid milk 20 ounces and corn syrup 1 ounce with caloric value of 21 calories per ounce, may be given. Or, half the concentration of an evaporated milk formula ordinarily used for an infant of the given age, with an ounce of rice flour added may be used.

Eiweissmilk or protein milk has wide acceptance in the treatment of this disease. If the diarrhoea has been severe, this feeding is especially indicated and 1 to 2 oz—6 or 10 times a day are given, increasing to a maximum of 40 ounces.

Great care must be exercised in changing to a cow's milk formula, and often we may need to keep the infant on evaporated milk for some time. The change to cow's milk is said to be facilitated by the addition of calcium caseinate—thus increasing the protein constituents of the whole cow's milk.

Drugs.—Drugs are of little value in the treatment of intestinal intoxication

in infants. Cathartics are early only irritating to the already inflamed mucous membrane. Later in the course of the illness, some infants who seem to be doing fairly well, still have 5-6 stools a day, and here perhaps opiates are indicated.

Many patients sooner or later show signs of collapse—and though stimulation is best effected by fluid administration — Mii of adrenalin 1:1000 hypodermically and perhaps caffeine may be given and repeated at short intervals.

Care of the mouth is very important in these infants also.

To control severe diarrhoea Bis-Sal. (gr. iii.) Tr. Cam. Co. (Min. iii.) and Chalk Mix. ad dr. i. may be used. Great disparity of view and conflict of theory still exist and the management of these cases is still extraordinarily difficult.

Moro's three words—toxicose, acidose, and exsicchose—will continue to remain the basis of rational treatment till we are able to control the disease by preventive measures.

Prevention And Public Health.

There is some evidence that infection by the pathogenic organisms implicated is also present in many other infants not ill enough to be hospitalized. Moreover in certain of the households which sent infants to the Hospital for Sick Children with intestinal intoxication, upon inquiry, one or more of the other members of the family may be found to be ill also, and stool cultures revealed similar organisms of a pathogenic nature to these isolated by infant stool cultures. It would seem, therefore, that the carrier state is apparent. It is highly probable that flies, etc., and carriers among the adult population serve to reinfect the younger generation. Some mothers are careless or uneducated in

the care of their baby or the preparation of its food. In such a situation infection by the mouth is easy and this is supported by the fact that only about 2% of cases occur in breast-fed infants. With the swinging of the pendulum back from the use of proprietary foods to mother's breast milk and with rational treatment one may expect a continued fall in the mortality rate in this disease.

It is true that in the twenty-five years from 1901 to 1926 the infant death-rate has fallen from 13.4 to 4.1, but still seventeen per cent. of deaths in the first year of life are due to diarrhoea and enteritis. The situation is largely a Public Health problem. Two features of infant life must be controlled if intestinal intoxication is to be eliminated—feeding and environment.

3T2 M.D.

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Notes on Investments.

By T. H. HODGSON, M.A., 3T2.

It is a sad commentary on the financial sagacity of the medical profession as a whole, that its members be placed high on the sucker lists of stock promoters and be considered as among the most gullible of candidates for get-rich-quick schemes. The reason for this is not hard to find. Probably no other profession absorbs so much of its members' thought and energy—certainly no other makes such demand upon their time—to the sacrifice of other causes. And it is part of human vanity that one who has spent many long years preparing for his profession, and many more thereafter to amass a principal, believes he can, without even an elementary knowledge of economics, consistently invest his earnings safely and wisely. For although very much time and thought are given to the making of money, little are given to its investment. It is in the best interests of every graduate that he formulate a definite investment policy as soon as possible.

Mortgages on real estate, preferred stock, common stock, and bonds constitute the chief investment classes. First mortgages are an excellent investment medium, but the mortgager must have an idea of the value of the real estate and also of the reliability of the mortgagee. But mortgages have no ready marketability and if disposed of before they are due go at a substantial discount.

Investment in the preferred stock of a company is not so very satisfactory, for this class has neither the security of a bond nor the speculative appeal of a common stock. The past decade has witnessed the rapid rise of common stock to the rank of the most popular investment medium. A variety of circumstances contributed to bringing this about. In the main, the rehabilitation following the world war, together with unprecedented advances in the realm of science and invention, so

stimulated a demand for goods of all descriptions as to greatly swell the profits of trade and industry. Holders of common stock in the fortunate companies saw their principal increase by leaps and bounds, and their dividends double, then triple, the original figures. At the same time holders of bonds in these same companies saw little or no increase in their capital investment, and none whatever in the interest they received. It is therefore little wonder that the public soured on the senior security, and that the almost random selection of common stocks appeared to be the highroad to wealth. Later, when the inevitable reaction had set in, holders of common stock saw their dividends vanish and their principal decimated. On the other hand, those who had foregone a chance of immediate profit in lieu of ultimate safety, and had retained their bonds, were in much the best position.

There are two classes of holders of common stock, and it is important to distinguish between them. One buys a security with the intent that it shall produce an income for him; the other buys with the idea of selling the security as soon as a reasonable profit can be made. The latter is only moderately concerned with the future prospects of the company or the dividend, if any, being paid, and is termed a speculator. The former is called an investor, however applicable either of these terms may be, since the activities of one may shade off into those of the other.

There has been, and still is, a strongly held idea that if one buys a security, puts it away in the strong box, disregards temporary fluctuations, literally forgets about it, he will come out ahead. Such an idea is utterly fallacious. Glance at the values and dividends of ten of the leading stocks in America of fifteen years ago, and compare with the prices and dividends

of the same stocks to-day. In the early years of the century railroad stocks and bonds were considered investments par excellence. Now, due in large measure to highway traffic and backward management, the railroads of America can barely meet their fixed charges. About the same time the stock of anthracite coal companies appeared invincible. But that was before the day of imported anthracite, of oil, and of natural gas. For several years the motor car industry, previously considered merely the "automobile game," enjoyed great prosperity, and for a somewhat shorter time the radio industry did likewise. The last group of stocks to hold the limelight has been the public utilities—the electric light, heat and power, the telephone, the gas, and the waterworks stocks. Here, the public reasoned, were companies that enjoyed a monopoly and furnished essentials, and that should earn money through good times and bad. But they failed to take into account the insistent clamor for public ownership of utilities, and the demand for lower rates. Truly there are fashions in stocks as well as in clothes. It cannot be denied that large fortunes have been made by holding a security for many years; but there are just as many cases in which the opposite result has obtained. It is the wise investor who regards none of his investments as being so good as not to require watching. He can then replace the wayward stocks with others for which the prospects are brighter.

It is obvious that it behoves every investor to diversify his holdings. One at times may be tempted to stake all in what appears to be a good thing, but it is less expensive to let discretion be the better part of valor, and forbear putting all the eggs in one basket.

If the holdings of the long-term investor require occasional scrutiny, those of the speculator need constant attention. The time so required is, in the latter case, obtained at the expense of the practice of one's profession. The

advice given to leave speculating severely alone, somewhat disqualified since it usually comes from one who has plunged and lost, and who will in all likelihood speculate again after he has recouped his losses, is nevertheless sound. For statistics show that only three out of one hundred who "play the market" over a period of years, come out ahead at the end of that time. A wider knowledge of the odds against him might deter the speculator, but this is doubtful, so great a hold has this type of gambling taken on the life of the nation. It is well to remember that in this game one is competing with the professional traders, and that the insiders will act on the information they get, long before the amateur has even heard it. In short, the dice are loaded.

All investors busily calculate the profit they expect to make on a security, but few stop to consider what they can afford to lose. It is the latter which is the really important thing. It is always a good plan to protect one's losses and to let the profits run on.

The selection of a security for investment is worthy of much thought. In the past the advice has been given to select the stocks of the largest units in the industry. These, however, have usually millions of shares of stock outstanding, dividends are small, and the yield is usually low on account of the inflated value placed upon the shares. There is increasing evidence that the big combines, with heavy overhead, unwieldy organizations, and plants geared for mass production, are having increasing difficulty in competing with the smaller, well-integrated units which can adapt their organization to local conditions. The outlook for these independents is thus better than it has been for some time past and they are worthy of investment consideration.

Much has been spoken and written against trading on margin, but there was one good thing about margin trading—the speculator was sold out if he failed to keep up his margin, and this turned out to be a blessing in disguise.

when the stock declined in value further. The greatest losses are taken by stubborn owners of stock who simply refuse to part with it because they have paid for it and feel they can hold it. It is a good thing to know when to stop, when to get out, in this as in other lines of endeavour. Commonly the holder of a poor stock which is dropping rapidly will sell one of his better stocks, or a bond, to protect it. This is unwise, and is sending good money after bad; strong holdings should never be sacrificed to protect weak ones.

The investor should obtain facts regarding the business in which he is interested and act on his own interpretation of them, rather than on the opinion expressed by another. The newspapers have been filled with predictions of better times by all and sundry for the past two years. Not long ago the president of a large Toronto business was reported in a newspaper interview as saying that his business was enjoying the best year in its history; shortly after that the firm failed. It is impossible to judge between what is sincere and offered in good faith, and what is false and told to further the best interests of the speaker. Statistics on freight car loadings, bank clearings, commodity and specialty sales, steel capacity, wholesale prices, etc., are published periodically and will

furnish any investor with an idea of the trend of industry. Figures never could lie like the contortionist.

The advantage of a little healthy pessimism might be conceded, because unlike unbounded optimism it would never have saddled the individual with more stock and the nation with more ambitious undertakings than either can find the money to pay for.

Bonds comprise the fourth group of security to be considered. The axiom that the backbone of every successful investor's portfolio should consist of seasoned bonds, though appearing a trifle homely a few years ago, was never so true as it is to-day. A good bond should combine safety of principal with a good yield over its term. Bonds of foreign countries, especially those politically unstable, should be avoided; the experience of investors in South American bonds has been sad. The bonds of the national government provide the best investment in the country. However, the ability to pay off bonds in this case depends on that to raise taxes, which in turn depends on the return from commerce and industry generally. Hence bonds are not a thing divorced from the current condition of industry. But if the federal government be forced to default on its bonds there will be little other security in the country worth more than the paper it is written on.

The Control of Coronary Circulation.

By D. R. E. MACLEOD, B. and M., '35.

The normal heart is capable of great adaptation to meet the varying need of the body. It is evident, then, that the coronary circulation is subject to accurate regulation so as to ensure a sufficient blood supply to the heart at all times. This control involves a number of factors, which can be conveniently divided into three groups: first, the mechanical or physical control; second,

the chemical control; third, the nervous control.

THE MECHANICAL CONTROL.

The mechanical control may be summed up in the term "pressure"; namely, pressure of the blood tending to distend the arteries, and pressure exerted by the heart muscle at each contraction tending to squeeze and close down the

vessels. It is important to remember that it is the interplay between these two antagonistic forces that determines the total flow through the coronary system.

Blood is supplied to the heart by two vessels, the right and left coronary arteries, which arise from the aortic sinuses. Blood is returned from the heart chiefly into its own auricle by venous channels, of which the largest is the coronary sinus. Other veins, notably the anterior cardiac veins and the Thebesian veins, open into the right atrium and the right and left ventricles. The Thebesian veins became of especial interest when Wearn, in 1927, as a result of perfusion experiments, stated that they communicate with the capillaries as well as with the coronary veins. He thought that there might also be a direct communication between the coronary arteries and the Thebesian vessels. Grant and Viko confirmed the first statement, but showed that direct arterio-Thebesian communications are anomalous. The importance of the venous anastomoses in the heart has been clearly demonstrated since, as Grant and Jones have shown, even the coronary sinus may be blocked without such interference to the coronary circulation as to occasion death. Even more interesting are the cases described by Wearn in which, though the coronary arteries had obviously been obstructed for some time, the individual was fairly active up till the time of death. These cases seem to prove that with the gradual occlusion of the arteries the Thebesian veins supplied blood to the capillaries.

Drainage Distribution.

The question as to the fraction of the blood drained by the coronary sinus was investigated by Evans and Starling. As a result of their experiments they found that the fraction drained by the coronary sinus was from 60 to 66% of the total venous return. Anrep and his collaborators confirmed these results and showed that the ratio was

only slightly affected by changes in aortic pressure, coronary pressure, rate or output of the heart. These findings were of importance since in subsequent experiments the blood flow from the sinus was taken as a measure of the total flow. They also found that the distribution of the blood between the arteries is relatively constant, the left coronary artery normally carrying from 80 to 85 per cent.

The Relation of the Coronary Circulation to the Different Phases of the Cardiac Cycle.

Some difference of opinion as to this question existed until Anrep and his co-workers performed a series of experiments in which the coronary inflow, coronary outflow and pressure changes of the ventricle were recorded simultaneously. The inflow and outflow were recorded by means of hot wire anemometers so that the record was in the form of a curve, the height of which represented the rate of flow. The sinus outflow curve shows three upward waves, representing three periods of increased outflow. The first accompanies auricular contraction and is due to it, since it follows any changes in auricular rhythm. The second, which is comparatively small, coincides with the period of isometric contraction. The third and largest wave begins with the period of ejection, reaches a maximum at about the end of ejection, and rapidly falls away. The periods of outflow accompany the contractions of the heart and are due to the squeezing out of the blood from the veins by the contracting muscle. Outflow ceases immediately after systole, since the emptied veins must first be filled again before any blood can flow from them. Anrep showed, by these experiments, that inflow is greatest during diastole and at the beginning of systole, being suddenly checked at systole due to the compression of the arterioles by the contracting muscle.

The Effect of Arterial Pressure.

The effect of increased arterial pressure, when the coronary perfusion pressure is kept constant, is a decrease in the coronary blood flow. The decrease is due to the increase in endocardiac pressure, which causes a more complete arresting of the flow during systole. In the body, however, it is always accompanied by a rise of pressure in the coronaries themselves. This more than counterbalances the former effect, and a rise in arterial pressure is always accompanied by an increase in the coronary blood flow.

The Relative Effect of Systolic and Diastolic Pressures.

The question may now be asked: "Does the amount of coronary flow depend more on the systolic or on the diastolic pressure?" This question is of some practical importance since we would like to know how the coronary flow is affected in conditions in which the pulse pressure changes without much change in the mean arterial pressure, as in aortic regurgitation, for example. Anrep and King investigated the question in the following manner: they varied the output or resistance so as to maintain constant either: (a) the systolic pressure; (b) the diastolic pressure; (c) the coronary blood flow. By their experiments they showed that the coronary blood flow is affected by changes in the systolic pressure as well as by changes in the diastolic pressure, and is not determined by either the systolic or diastolic pressure separately, but closely follows variations in the mean arterial pressure. By these experiments they also showed that changes in the output cause little or no change in the coronary flow.

The Effect of Rate.

In a similar series of experiments, in which the output and the rate were kept constant, Anrep and Kind showed that heart rate, in itself, has no effect upon the coronary flow. In a later paper Anrep and Häusler showed that,

other factors being constant, an increase of rate tends to decrease the coronary blood flow slightly. This is due to the greater relative shortening of diastole, during which phase the major part of the flow takes place. On the other hand, with an increase in rate, the contractions may become less powerful, and may not arrest the flow as completely as the more vigorous contractions of the better filled heart. The first factor, as a rule, slightly overbalances the second, the net result being a small decrease in flow with increase in rate.

The Effect of Fibrillation.

In the same paper, Anrep and Häusler stated that auricular fibrillation has no effect on the coronary circulation, but in ventricular fibrillation there is an increase due to the removal of the restricting effect of systole.

THE CHEMICAL CONTROL.

The Effect of Oxygen Saturation.—

That the oxygen saturation of the blood is an important factor in regulating the coronary flow has been demonstrated by Markwalder, Starling and others. Hilton and Eichholtz later conducted experiments in which artificial respiration was maintained and the oxygen saturation of the blood decreased by substituting nitrogen for air. When the oxygen saturation of the arterial blood decreases there is a great increase in the coronary flow, which keeps pace with, and compensates for, the diminished oxygen saturation. This is shown by calculation of the oxygen consumption of the heart during the same period. There is no evidence of oxygen lack during anoxemia since the oxygen consumption of the heart actually shows a slight increase. This seems to show that the dilatation is due to a direct reaction of the arterial wall to the diminished oxygen tension of the blood, and is not due to an accumulation of metabolites on account of oxygen lack.

The Effect of Carbon Dioxide and Reaction of the Blood.—It was observed by Hilton and Eichholtz that dilatation of the coronary vessels and increased flow was produced by the addition of carbon dioxide to the inspired air, though the effect was much less than that caused by oxygen lack. They found that lactic acid similarly produced dilatation, but that the change in Ph was only minimal, being from 7.8 to 7.7. With carbon dioxide the Ph changed from 7.7 to 7.4. It is probable that the hydrogen ion concentration is the important factor, but this point has not been definitely proven.

The Effect of Drugs.—When a drug is administered it must be remembered that it may affect the coronary flow directly by vasodilatation or contraction, or indirectly by changing the rate or strength of the heart beat. The direct and indirect effects may be antagonistic or they may be additive, depending on the nature of the drug. The rate of flow during diastole is dependent upon the state of dilatation of the coronary arteries, while the rate of flow during systole is determined by the strength of the contractions. Thus an increase in the rate of flow during diastole indicates vasodilatation, and vice versa, while an increase in the systolic flow shows a weakening of the heart beat.

Amyl Nitrite.—When amyl nitrite is administered the rate of flow during diastole increases. No change occurs during systole. This shows that this drug has no effect on the heart beat but increases the coronary flow by vasodilatation.

Adrenalin.—Most observers have reported an increase in the coronary flow on administration of adrenalin, but some have found a decrease. This latter effect was obtained because they injected an amount which was insufficient to cause any vasodilatation but which did increase the rate and strength of the contractions. These factors

caused a decrease in the flow. With larger doses a preliminary diminution is observed followed by a definite increase in the flow. The initial decrease is caused by increase in the strength and rate of the beat, which begins before any direct effect on the arteries occurs. Later, vasodilatation more than off-sets this and the flow is increased.

Pituitary Extract.—This drug has no effect on the heart beat in small doses, but causes vaso-constriction of the coronaries.

Carbon Dioxide.—The increase of flow produced by carbon dioxide is due to weakening of the heart and to vasodilatation.

Histamine exhibits a species difference since it causes dilatation of the coronaries in the cat but constriction in the rabbit and dog. Cruickshank states that it causes dilatation of the isolated human coronary artery and its action in the body is no doubt the same.

Digitalis and Caffeine.—These drugs strengthen the heart beat and increase the coronary blood flow.

THE NERVOUS CONTROL.

The coronary arteries were shown by Wallard to receive a double innervation. The larger branches are supplied by both vagal and sympathetic fibres, while the smallest branches receive only vagal endings. No nerve supply to the capillaries has been found. Stimulation of the vagus has been shown by a number of workers to cause a constriction of the coronary vessels and a decrease in the coronary flow, showing the presence of vasoconstrictor fibres. The vasoconstrictor fibres differ from the cardiaco-inhibitory fibres in that they are less excitable. A weak stimulus may cause a slowing of the heart rate but no decrease in the coronary flow. Administration of small doses of atropine abolishes the cardiaco-inhibitory effect of the vagus, but does not paralyze the

vasoconstrictor fibres. The latter also exhibit a longer latent period. The vagal fibres possess tone. This was shown by Anrep and Segall, who demonstrated an increase in the coronary flow on section of the vagi.

The innervated heart differs from the denervated heart in that it responds to changes in the output. This is believed by Anrep and Segall to be brought about by a coronary reflex by which vagal tone is diminished. They consider it to be a proprioceptive reflex similar to the Bainbridge reflex, but independent of this effect.

Stimulation of the stellate ganglion increases the coronary flow, showing the vasodilatator action of the sympathetic fibres. They do not possess tone, since extirpation of the stellate ganglion has no effect on the coronary blood flow. Variations in the cerebral blood pressure affect not only the general arterial blood pressure, but also the coronary circulation, an increase of cerebral pressure causing vasoconstriction. This regulation is brought about in part by the carotid sinus reflex. Whether or not it is entirely due to this reflex has not been definitely proven.

In general, it may be said that each cardiac reflex is accompanied by a coronary reflex, so that any increase in the work performed by the heart causes a corresponding increase in the coronary blood flow. In the body the nervous control, together with the oxygen content of the blood, is of chief import-

ance in regulating the coronary flow. It acts rapidly and brings about the required change before any conspicuous alteration in arterial or venous pressure occurs. The control of the coronary circulation affords us another example of a sensitive mechanism bringing about a rapid and accurate adjustment to meet the varying conditions imposed upon it.

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The Treatment of Varicose Veins.

By J. H. COUCH, M.B., F.R.C.S. (Edin.), Department of Surgery.

The treatment of varicose veins has been revolutionized during recent years. Only a short time ago the regular treatment was by operation. A tedious dissection was involved, and long, unsightly scars remained at the various points of incision between the upper thigh and ankle. Nor were the final results satisfactory. Aside from expense and tedium of a convalescence lasting from four to six weeks, the enlarged veins themselves recurred in 15% of cases, and approximately $\frac{1}{2}$ % of all patients subjected to operation died of an embolus to the lung.

Varicose veins may now be completely cured without interfering with the patient's daily routine of work. The operation involves nothing more than a needle prick, no scars are left, about 4% only recur, and the number who die from pulmonary emboli is only 1-70th as great as under the former treatment. In fact, the danger of emboli is less after injection than in cases left untreated.

A brief description of the venous circulation in the leg is necessary if the treatment is to be understood. The muscles of the leg are ensheathed in a strong, unyielding covering of deep fascia within which are large veins, well supported by muscles, capable of enormous work and never subject to varicosities. It is these veins which are damaged in "milk leg," or any form of deep thrombophlebitis. Under normal conditions, the work of these deep veins consists of the return of the blood from the muscles and bones. On the outside of the deep fascia is another set of veins lying in the fat and superficial fascia under the skin. Their function, normally, is to return the blood from the skin and superficial fascia, emptying it into the deep veins in the groin.

Varicose veins are not produced by any single cause, but in all cases several factors are at work. If an indivi-

dual with inherited poor veins be further debilitated by illness, and then be subjected to circumstances which harass the return of blood from the leg, varicosities of the superficial veins may be caused. Certain occupations which require constant standing are precipitating factors, as are also conditions which cause increased intrapelvic pressure, such as pregnancy.

Once a slight dilatation has occurred the valve cusps no longer meet in the centre of the vein, and blood leaks past them. Normally no valves are present in the intra-abdominal or thoracic veins. Therefore, these superficial unsupported veins are required to return a column of blood extending from the foot to the heart, representing a considerable lift against gravity in the erect position. Gravity overcomes the propulsive force, the flow is reversed, the vein below dilates and stretches, and a typical tortuous varicose vein results. At the saphenous opening, some blood overflows back down into the long saphenous vein and a vicious circle is established. Thus varicose veins are worse than useless, because not only do they fail to do their own work, but also add to the burden of the deep veins. The rationale of improving return circulation by obliterating veins is based on this fact.

The following are the more important contra-indications to the injection treatment of veins. Certain conditions, such as deep thrombophlebitis, following pregnancy, typhoid fever or operation, render permanent damage to the deep veins, and thus the enlarged superficial veins must never be injected. Of course, in such cases it will be noted that the direction of flow is upward, and not distally as in the uncomplicated varicose veins. Acute or subsiding phlebitis, evidenced by the typical signs of inflammation, warn of the danger of a widespread extension of the process. The occasional bacter-

aemia accompanying boils or acute sore throat predisposes to a localized suppurative process at the site of injection.

The injection is carried out as follows. The patient is seated on a table with the foot resting on a bench below while the operator sits in front. A small glass syringe with a 24 gauge long hypodermic needle, having a short bevel and a sharp point, is introduced, bevel down, into the vein. When blood is seen, the vein is stripped empty above and below by the thumb and first finger, and injection made rather quickly. One cubic centimetre of 5% sodium morrhuate is injected in each of two places at each sitting. These two places are usually about four inches apart and cut off a segment of the same vein. Immediately on withdrawing the needle the vein is compressed at the site of puncture by means of a small pledget of cotton held firmly in position with a strip of adhesive for 48 hours. The patients are encouraged to continue their ordinary duties. Such injections may be repeated once or twice weekly, and the average case requires five sittings.

The primary object is to produce a severe chemical damage of the intimal lining of an empty vein, in order that a sterile inflammation may be caused and the two walls of the vein fused together. The injection of the irritant chemical causes the vein to go into spasm which contracts it to a small size, and the thrombus which does result is therefore small and firmly adherent to the vein wall. That emboli are so rare is due to the fact that the reverse flow of blood tends to jam the clot tighter into the vein rather than to wash it into the general circulation.

Sometimes a slight brownish discoloration appears over the hard vein, and occasionally the reaction extends out for one inch on either side of the involved vessel. Thus a feeling of stiffness and soreness and a tender hard mass may cause some discomfort for 48 hours. All traces of the vein will disappear in about six months.

Occasional untoward effects may follow as a result of individual idiosyncrasy or errors in technique. Cramps in the muscles of the calf are encountered, but were more usual when strong saline solutions were used. Local pain may occur when some of the solution is injected outside of the vein wall. Toxic manifestations were more usual when quinine injections were used, and consisted of headache, tinnitus, uterine contractions or urticaria.

Later complications may occur some time after injection. Periphlebitis, with a widespread, hard, red, tender swelling, necessitates bed and cold saturated magnesium sulphate compresses for five or six days. Slough from the injection of a strong irritant outside of the vein wall may cause an indolent ulcer, best treated by complete incision and suturing of the edges. A haematoma may result at the site of injection. Pulmonary embolism has been reported and is a dangerous complication, but fortunately very rare.

Varicose veins if untreated always tend to become steadily worse, and, over a period of years, result in cellulitis and indolent ulcer. The treatment of such complications necessitates, besides vein injection, such procedures as Unna's paste stocking, division of the saphenous nerve, lumbar sympathectomy, and surgical excision, with possible skin grafting.

A MEDICAL DETECTIVE STORY.

The following story is told by Grant Irving of 3T2, of a prominent London physician, Dr. Donald Hunter, co-author of "Clinical Methods."

Patient, William Stokes, English, short, corpulent, rugged physique; occupation, farmer. He appeared at the Out-patient department of London Hospital with vague gastric symptoms, was given some medicine, and went back home. Six months later, he appeared at the Hospital with wrist-drop, foot-drop, black gum-line, and severe colic—the typical picture of lead poisoning.

But "Old Bill" was a simple farmer, who all his life had breathed the fresh, pure air of Devonshire moors. He had never been near a garage with batteries, nor had his house any piping of any kind. His diet appeared blameless; he was not a painter. Whence came the lead? Dr. Hunter had given up the problem, when a week later, on reading over some old journals, he came across a description by a Dr. Miller in 1781, of lead poisoning from Devonshire cider. The pressing blocks of the cider-mill had been covered with lead. Ten minutes later it was ascertained from a blushing William that "He loiked his soyder, and the 'arder the better."

The next day the Doctor was in Devon. But the condition of the presses had been remedied a century back, and the brewery owners, incensed at the reflection on their product, berated the chagrined physician in no uncertain terms.

Still smarting from the brewer's remarks, the place where William bought his cider was sought so as not to arouse suspicion. During the noon-hour rush at Bill's favorite pub, a sample of the beverage was obtained. Again disappointment,—for the chemical test for lead was negative.

On the way to the train, the amateur detective called in for a chat with the bartender.

"By the way," he said, sipping a

glass. "Did Bill Stokes seem to like this cider?"

The bartender chuckled: "Loike it? Gor blimey 'e was waitin' at the door every mornin' for the first glass out of the tap!"

"Oh! By the way, how does the cider reach the tap here?"

"We pipe it from casks in the cellar."

A pound note flashed over the bar to quell all opposition, and in two jumps the doctor was in the cellar. There were the casks and, sure enough, away in the dark corner was a lead pipe leading up to the tap at the bar. The mystery was solved. The hard cider contained a goodly amount of acetic acid, and William, getting the first glass in the morning, received a full dose of the lead acetate which formed in the cider lying in the pipe all night.

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